Case Report

Progressive flaccid paraparesis with albuminocytologic dissociation: It's not always Gullain-Barre syndrome

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Context: Spondylodiscitis, or vertebral osteomyelitis, is an unusual infection of the vertebral bodies and intervertebral discs that can occasionally present with neurological signs.
Findings: We present a patient with subacute flaccid paraparesis with associated albuminocytologic dissociation who was eventually diagnosed with spondylodiscitis.
Conclusion: The case presented depicts a diagnostic difficulty encountered in clinical practice: Albuminocytologic dissociation in CSF is not always attributed to Guillain-Barre syndrome and other possible causes such as obstructive spinal cord lesions must always be considered.

KEYWORDS: Spondylodiscitis, Vertebral osteomyelitis, Flaccid paraparesis, Albuminocytologic dissociation

Introduction

Spondylodiscitis, or vertebral osteomyelitis, is an unusual, with estimated incidence of 2.4 patients per 100.000,[1] infection of the vertebral bodies and intervertebral discs which may occasionally present with neurological signs and symptoms. The most common pathogens responsible for this entity are Gram positive bacilli (Staphylococcus Aureus, Streptococcus) as well as Gram negative aerobic bacilli (Escherichia Coli, Pseudomonadas Aeroginosa, Proteus).[2–4] We present a patient with subacute flaccid paraparesis with associated autonomic disturbances that was eventually diagnosed with spondylodiscitis, although his clinical condition was highly suggestive of Guillain-Barre syndrome.

Case presentation

A 57-year old man with a history of diabetes mellitus presented to the emergency department complaining about progressive walking difficulty which started a few days ago associated with urinary retention and constipation and back pain that begun 10 days earlier. Two months ago, he was hospitalized in another institution due to epidermal abscess located in the lower back, which was treated with local drainage and antibiotics. At that time he did not report any other symptoms than lower back pain which resolved completely after treatment. His rest medical and family history were unremarkable.

Neurological examination revealed flaccid paraparesis (MRC grade 4 in both lower limbs). Although he was complaining about paresthesias in both lower limbs, pain and temperature sensation were intact. No sensory level was objectively recognized. ASIA Impairment Scale (AIS) was found grade D (incomplete injury with neurological level of injury on L2) and International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) subscores were UEMS total 50/50, LEMS total 40/50, LT total 112/112 and PP total 112/112. The patient also mentioned significant weight loss during the past month (about 10 kg). He denied any headaches, visual abnormalities and he did not demonstrate any cognitive decline. Fever was absent. He was hospitalized for further evaluation.

Initial brain CT was normal. His routine work up revealed mild elevation in neutrophils and ESR (102 mm/h) but was otherwise normal. CSF examination was acellular with normal glucose levels but showed a significant increase in total protein (332 mg/dL), findings suggestive of albuminocytologic dissociation.

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No infectious agents (including herpes simplex virus with PCR) were identified. Immunological tests (ANA, ENA, anti-dsDNA, anti-Ro, anti-La, RF) serum B12, copper and ACE were negative. Nerve conduction study showed mild prolongation of F-wave latency in both lower limbs.

Gullain-Barre syndrome was suspected at this point and the patient received intravenous immunoglobin 0.4 g/kg/day for 5 days. Despite treatment his clinical condition continued to deteriorate. Given the fact of persistent lower back pain and lack of clinical improvement after administration of immunoglobulin, MRI of the spine was performed, which showed high T2 signal of the spinal cord at T9-T10 level and paravertebral abscesses which demonstrated high signal on DWI sequences. Additionally subsidence of the T10 vertebral body associated with bony fragment compressing the spinal cord was found (Fig 1). Tumor markers and Vidal-Wright tests were negative as well as testing for Mycobacterium Tuberculosis. Blood culture was positive for Staphylococcus Aureus and the patient received antibiotic treatment with intravenous linezolide 600 mg twice daily for 15 days followed by oral linezolide 600 mg twice daily for 2 months. On follow-up 3 months later he was able to walk unassisted and his back pain was significantly improved.

Discussion

Spondylodiscitis may be acute, subacute or chronic. Its estimated incidence is 2.4 per 100.000.[1] It is more common in the elderly and immunocompromised patients, such as diabetics or those receiving corticosteroids. Infection of the vertebral bodies and discs may result from hematogenous spreading, spinal surgery or from adjacent tissue infection.[2] Staphylococcus Aureus is the most common pathogen, followed by Escherichia coli.[3–6] However other infectious agents have been associated with spondylodiscitis such as Proteus Mirabillis and Mycobacterium tuberculosis, with the latter causing lesions on multiple vertebral segments.[7]

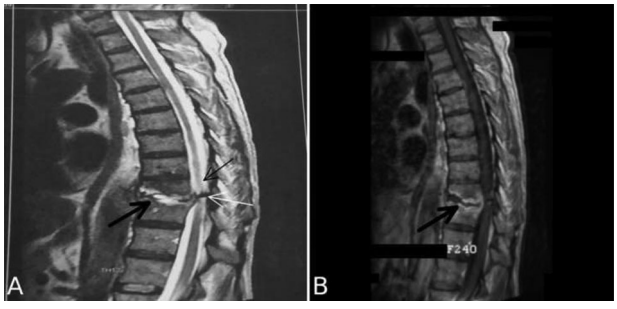

Fig 1 a) MRI T2 sequence of the thoracic spine, showing high spinal cord signal in the T9-T10 level suggestive of myelopathy (thin black arrow) and subsidence of the T10 vertebral body due to spondylodiscitis (thick black arrow). Also note a bony fragment causing spinal cord compression and obstruction of CSF flow, leading to raised CSF protein levels (white arrow). b) T1 post Gd+ showing gadolinium enchancement and destruction of the T10 vertebral body (black arrow).

Back pain is the most common symptom of spondylodiscitis in approximately 80% of cases, followed by fever (35-60%). Spondylodiscitis may be associated with neurologic complications in one third of patients.[3] These usually appear in the form of radiculopathy or myelopathy, resulting in weakness, sensory and autonomic disfunction. Since spondylodiscitis may be a complication from a distant site infection, symptoms from other systems may predominate.

Initial routine blood work-up is helpful, as it can demonstrate a rise in neutrophil levels, ESR, and CRP. However in rare cases, initial laboratory testing may be entirely normal. Plain radiography is helpful as a first step, since it is widely available and may reveal an alternative diagnosis (e.g. bone metastases or an osteoporotic fracture). However its sensitivity in detecting spondylodiscitis is relatively low. MRI of the spine is the modality of choice, demonstrating high signal changes at the disks and vertebral bodies in T2 sequences.[8] Caution is warranted in cases of erosive osteochondritis, as this disease may have similar appearance in MRI.[9] Biopsy of the lesion is indicated in cases of diagnostic uncertainty.

Targeted antibiotic therapy given intravenously is the basic pillar of treatment. The regimen selected must target the responsible pathogen that has been isolated from blood cultures.[10] In rare cases, when no particular microorganisms have been identified, empirical antimicrobial therapy is initiated. The recommended duration of therapy varies from 4-6 weeks to 3 months.[11,12] Surgical intervention is restricted in cases of spondylodiskitis with associated abscesses.[10]

The case presented depicts a diagnostic difficulty encountered in clinical practice: The nonspecific elevation of total protein in CSF in cases where its flow is obstructed by spinal cord lesions. Initially, due to this finding, Gullain-Barre syndrome was considered as the probable diagnosis. However clinicians must be cautious in order to properly evaluate cytoalbuminologic dissociation. This can reduce diagnostic errors and prevent delay of targeted treatment. When a patient, especially if he is immunocompromised or diabetic, presents with a subacute paraparesis and bladder and bowel dysfunction, associated with back pain, spondylodiscitis must be considered in the differential diagnosis.

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Disclaimer statements

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Ethics approval None.

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